THE RHODE ISLAND MEDICAL JOURNAL

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VOLUME XV
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PROVIDENCE, R. I., OCTOBER, 1932
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by HARRY C. MESSINGER, M. D.

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ENTERED AS SECOND-CLASS MATTER AT THE POST OFFICE AT PROVIDENCE, R. I., UNDER ACT OF MARCH 3, 1879

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ORIGINAL ARTICLES

SOME SIDELIGHTS IN THE BIOGRAPHY OF MY GREAT, GREAT GRANDFATHER, CALEB FISKE, M.D., ARMY SURGEON, PHYSICIAN, JURIST, FINANCIER*

By LOUISA PAINE TINGLEY, M.D., F.A.C.S.
PROVIDENCE, R. I.

Each year the Trustees of the Fiske Fund offer a premium for the best essay on a given subject, which they may select.

I wonder how many of the physicians taking part in this competition consider or know anything about the man who made this prize possible? He was a man of vision and learning, and desirous of handing down to others the inspiration for knowledge and research which he, himself, had received. I look upon this prize as a challenge to the members of the Rhode Island Medical Society to carry on in the advancement of medical education, following the example of Dr. Fiske.

In 1834 the Society received by the will of Dr. Fiske $2,000 for the establishment of the Fiske Fund. This amount has been largely increased, the income being used for the payment of the premiums.

He also left to the Society a large portion of his medical library, which I have been unable to identify.

A quotation from the will of Dr. Caleb Fiske furnishes his conception of the uses for which he desired his legacy to the Rhode Island Medical Society to be put.

"I give and bequeath to the President and two Vice-Presidents of the Medical Society of the State of Rhode Island, for the time being and to their Successors in Office the semi-annual dividends arising from Forty shares of Stock, which I own in the Union Bank, in Providence, the amount thereof *Two Thousand Dollars,* in *Trust* for the uses herein limited. Use the first, *Nine twelfths* of said dividends shall institute a *fund* to be applied in the manner following, to wit, the said Trustees or either two of them, shall select at every annual meeting of said Society, such Subject or Subjects for investigation as they may judge most conducive to the advancement of Medical Science, and give notice thereof in one of the Newspapers published in Newport, and in one published in Providence for the term of six weeks, offering such premium or premiums as the annual product of said fund will justify, to be awarded and paid by said Trustees for the best Treatise on the subject proposed by them for investigation, to be communicated to said Trustees one month previous to the next annual meeting of said Society. And in order that a laudable spirit of emulation may be excited and maintained, the Trustees shall not only suitably reward the Authors of the fortunate productions, but also prescribe such rules for receiving the communications and deciding on the merits of the several performances as will shield the unsuccessful competitor from obliquy or reproach. *Use the second, Two twelfths* of the profits or dividends of said Stock is to remunerate said Trustees for their services in the execution of the several Trusts herein confided to them. *Use the third, One Twelfth,* of the profits of said Stock is to be appropriated to *printing* and *supplying* each member of said Society with a copy of such Treatise for which premiums shall have been awarded. It is believed, however, that the copy right of those productions may be so disposed of by said Trustees, as not only to furnish the Members of said Society with copies gratis, but also make some addition to the aforesaid fund. And it is also believed that said Trustees, in consideration of the advantages which said Society may derive from a discreet management of said fund, will frequently if not *uniformly,* render their services *gratuitously,* whereby a further addition may be made to said fund, if these anticipations should be realized in part or in whole, whatsoever sums may remain unappropriated, shall be added to said fund for the uses aforesaid. And my will further is, and I hereby ordain, that the aforesaid *Forty Shares of Stock,* the nominal amount whereof is *Two Thousand Dollars,* shall remain registered in my name in the books of said Bank, but the dividends or profits arising therefrom, shall be subject from time to time to the orders of said Trustees, for the uses above limited, but every order drawn on said Bank shall express the uses to which the money is to be applied. Furthermore, the said Trustees shall cause their proceedings in the premises to be recorded in a Book or Books from year to year, and deposited in the *Archives* of said *Society for safe keeping,* inserting therein the annual amount of said fund, and the addition, if any, made thereto, the subject or subjects proposed for investigation, the amount of premiums offered, the names and places of abode of the persons to whom premiums are awarded, with such other facts and remarks relative to the application of said dividends, as they may judge expedient, and the proceedings, or such part thereof as shall have accrued

*Paper read before the Rhode Island Medical Society at the Annual Meeting, June 2, 1932.

each preceding year, shall be audibly read before said Society at their annual meeting, and also be subject to the inspection of any members of said Society, and also be free for examination by *my heirs at law.* Provided, nevertheless, and I do hereby ordain that if the said Dividends or profits arising from the before named Stock or any part thereof, should at any time hereafter *be used for any other purposes* than those before limited, or applied in any other manner than is above directed, or if said Trustees or either two of them for the time being, should neglect or refuse to execute the aforenamed Trust, in manner and form above specified, (sickness and other unavoidable incidents excepted) for the term of one year, then and in either of those cases, this bequest shall thenceforth cease and determine, and the said Stock and the dividends arising therefrom, shall thereupon descend to and vest in *my heirs at law.* Provided, also, and I do hereby ordain, that if at any time hereafter said Medical Society shall discontinue its anniversary meetings authorized by its Charter, or in case its members (fellows) exclusive of Honorary members, should decline in number, and at any time hereafter be reduced to twenty, then and in either of those cases this bequest shall thenceforth cease and determine, and the said Stock and the dividends or profits arising therefrom shall descend to and vest in *my heirs at law.*"

The Fiske Fund has produced many prize essays, about half of which have been within Rhode Island. The first award of $40, given in *1835* to Thomas H. Webb, subject: "What are the Causes and Nature of Rheumatism and the Best Mode of Treatment to be Employed Therein?"

The same year, David King, M.D., was awarded $40. His essay was entitled, "What are the Causes and Nature of Purpura Hemorrhagica and the Best Mode of Treatment to be Employed Therein?"

In 1844 an award of $50 was given to Joshua B. Chapin, M.D., "Tenotomy: Its Comparative Advantages and Disadvantages."

In 1854 the award was $100.

In 1858 the award was $200.

In 1931 the award of $200 was given to Albert H. Miller, M.D., a member of the Rhode Island Medical Society, for his most complete and scholarly paper on "Anaesthesia."

The premium of $200 offered this year by the Trustees of the Fund should be productive of an instructive contribution for both ophthalmologists and internist alike, "The Value of Ocular Signs and Symptoms in the Diagnosis of General Disease," holds such an important place in the practice of medicine today.

Dr. Fiske is named in the Act of Incorporation of the Rhode Island Medical Society as one of its original members.

I have his Certificate of Membership in the Society, dated April 29, 1818.

Signed by JOHN W. RICHMOND, *Clerk*
PARDON BOWEN, *President.*

Dr. Fiske was the second vice-president of the Society from 1815 to 1818; the first vice-president from 1818 to 1823; and its president from 1823 to 1824.

Caleb Fiske, M.D., was a direct descendant of Robert Fiske of Laxfield, County Suffolk, England, 1525-1600.

He, in turn, was a great, great grandson of Symond Fisk, Lord of the Manor of Stadburgh, Laxfield Parish, England, 1300 to 1422, Symond was grandson of Daniel Fisc of Laxfield, County Suffolk, England, in the reign of King John, 1200. Daniel received a grant of land in Digneveton Park, from the Duke of Loraine, a title of which is in the Public Record of London, dated May 1, 1208. The Coat of Arms and Charter were granted by the Herald's College, and are registered in the Heraldry Gazette of Middlesex County, England. The Latin inscription thereon, translated, reads:

"Increase in Virtue, such is the Way to Immortality."

Caleb Fiske was the son of John Fiske of Scituate, Rhode Island, and Elizabeth Williams (who was a great granddaughter of Roger Williams).

He was descended in the paternal line from an English ancestor, who migrated to Plymouth, Mass., in December, A.D. 1620, and is of the fifth generation.

He was born in Scituate on Wednesday, the 24th day of January, A.D. 1753, in the house built by his grandfather, Benjamin Fiske, in 1727. He married Mary Manchester, daughter of Thomas Manchester, on Sunday, the 24th day of June, A.D. 1776, ten days previous to the Declaration of American Independence.

They had six children, one son, Philip, and five daughters. Thirteen grandchildren. Many great grandchildren.

Nabby, his first child, died when she was three months old of small pox.

Eliza died in her 20th year of phthisis pulmonalis.

Harriet Fiske Rae died in her 28th year of pulmonary consumption, "so fatal to the fair sex. This excellent woman whose early exit wrung the hearts of her friends with poignant grief was eminently distinguished for her mental Powers, engaging manners and signal Virtues." "The life is long that answers life's great end."

"She left an infant son, Caleb Fiske Rae, Born Jan. 29, 1810." (He died in Stockton, California, January 10, 1881, aged 70 years and 11 mos.)

Abby Fiske, a daughter, had one child, Oceana Harris, who died a consumptive in her 13th year.

Philip Manchester Fiske, Dr. Fiske's only son, died Jan. 31, 1828, of angina pectoris, in the 46th year of his age.

Mary Fiske, wife of Calef Fiske, died November 1, 1918.

The above data was copied from Dr. Fiske's family Bible, and was written in his own handwriting.

Tuberculosis claimed most of the women of his family in their youth.

Dr. Caleb Fiske died in Scituate, his birth place, October 4, 1834, aged 81 years and 8 months. He left a lengthy and descriptive will written in his own handwriting, dated March 12, 1831. Codicil dated July 15, 1834.

On a tombstone in the old family burying ground in the rear of his house in Scituate, R. I., is found the following inscription:

CALEB FISKE

DIED OCT. 2, 1834. AGED 82 YEARS.

Doctor Fiske served as hospital surgeon in Gen. Sullivan's expedition against Rhode Island.

Soon after the Revolution he was elected Judge of the Common Pleas. In 1823 he was chosen President of the Rhode Island Medical Society, which office he held for several years.

He passed an active, vigorous and discriminating round and was distinguished for his professional industry and extensive medical attainments.

His protracted life and the extended sphere of his practice gave him large personal acquaintances whose regard he secured by his affable manners, strict integrity and *sound morality*. Mary, wife of Caleb, died Nov. 1, 1817, aged 64 years.

Dr. Fiske's professional studies were pursued under the direction of Dr. William Bowen, one of the most extensive and successful practitioners of his time, and the instructor of a number of the ablest physicians of Rhode Island.

The general practice in the education of physicians prior to the organization of the Medical Society in 1812 was for pupils to enter their names as apprentices, in some physician's office. The teacher gave the pupil a letter of recommendation on leaving, which was his only credential.

After the society was organized, a few pupils were examined and licensed by its censors. In 1800 there were but five medical graduates in Rhode Island. Dr. Fiske, in addition to the cares of an extensive practice, became the teacher of some of the most distinguished doctors of the State. Among them are found the names of Drs. Niles Manchester, of Pawtucket; Daniel Baker of Cranston; Dr. Harding Harris, and the following gentlemen: Drs. Rowland Greene of Cranston, Stephen Harris of Providence, and Daniel Greene of East Greenwich.

The Medical School of Brown University was established in 1810. The rank which Dr. Fiske held among the physicians of the State led the University in 1821 to confer on him the honorary degree of Doctor of Medicine. In the cause of medical education in Rhode Island he took a deep interest.

Having received his certificate of qualification as a physician, he offered his services to the government, to act as a surgeon in the Army. In June, 1789, he received his commission as surgeon, Third Battalion Militia, Providence County, and served in General Sullivan's expedition against the British on Rhode Island. In 1795 he received his commission as captain in a Scituate company.

On the completion of his term of service he returned to his native place, where he practiced his profession during his long life, being, at the time of his death, the oldest practitioner in the State. He was often called in consultation by physicians in Providence, Newport, and other cities.

Family tradition states that Dr. Fiske, during the later years of his life, met with an accident, sustaining permanent injury to his knees, which made it impossible for him to walk. After that he often remained in his chaise, having patients brought to him for consultation and treatment.

In May, 1781, Dr. Fiske was appointed Fifth Justice of the Court of Common Pleas, for Providence County.

In May, 1781, he was appointed Fourth Justice of the Inferior Court of Common Pleas, for Providence County, in room of Caleb Aldrich, who is advanced.

In May, 1782, he was appointed Fourth Justice of the Court of Common Pleas for Providence County.

{ HERBERT O. BRIGHAM
From
State Record Commission }

It was said of Dr. Fiske "that he was versed in law as well as in physics. I think I have heard too of his wearing the ermine with as much grace as he was wont to handle the scalpel."

"Dr. Fiske was a man of distinction in the town, living on Bald Hill, at the southeast part of the town. In 1781 and 1783 he served as 'Moderator of the town meeting.'"

MRS. ELIZA FISKE

TO DAVID COLVIN, JR., DR.

1831				
April 11—	8 lb. Veal	6	.....	$ .48
" 26—	11 lb. Veal	5½	.....	.60
May 5—	7 lb. Veal	5	.....	.45
" 10—	10 lb. Veal	5	.....	.50
" 20—	One peck Quohogs		.....	.14
	10½ lb. Veal	5	.....	.53
	42 lb. of Beef		.....	.34
				$3.04

Received payment in full
DAVID COLVIN, JR.

The above bill is a copy of one found among Dr. Fiske's papers.

I have in my possession the copy of a bill:

ESTATE OF MRS. ELIZABETH CUMBERLIN
TO CALEB FISKE, DR.

(The dates of visits are given from June 17, 1829, to August 27, 1830.)

To Sundry Visits, Consultation, Advice, Attendance, Medicines, Directions and for the said Elizabeth in her life time being 118 Entries per book	$108.71
To Cash paid Mr. Eaton for two Bottles of his medicine	1.75
	$110.46

1830 September 25th, Rec'd the above Sum of One Hundred & Ten Dollars 46/100 of Eliza A. Fiske in full payment of the above amount.

CALEB FISKE.

Items from another bill make the charge on one occasion of fifty cents for "blood letting" and another for "consultation" with Dr. Bowen of fifty cents.

CERTIFICATE of a Carriage, chargeable with the yearly rate of Two dollars duty.

No. 276.

THIS IS TO CERTIFY, that Caleb Fiske of the town of Scituate in the County of Providence, in the second Collection District of Rhode Island, has paid the duty of Two dollars, for the year, to end on the thirty-first day of December next, for and upon a Two Wheel Carriage for the conveyance of persons, called a Chaise owned by Caleb Fiske.

THIS CERTIFICATE to be of no avail any longer than the aforesaid carriage shall be owned by the said Caleb Fiske unless said Certificate shall be produced to the Collector by whom it was granted, and an entry be made thereon, specifying the name of the then owner of said Carriage, and the time when they became possessed thereof.

GIVEN in conformity with an act of Congress of the United States, passed on the 24th day of July, 1813.

WILLIAM B. MARTIN

Collector of the Revenue for the second Collection District of Rhode Island.

Providence, July 24th, 1814.

The village of Fiskeville, Rhode Island, was named for Dr. Fiske. He and his son, Philip Manchester Fiske, built a mill and established the cotton manufactory business there and carried it on extensively for many years. This mill has since burned.

They also built the village and mills at Jackson, which were subsequently sold to Gov. Charles Jackson, after whom the place was named.

The house and grounds owned by Dr. Fiske's son, Philip, in Fiskeville, as a manufacturer, are now used for the annual fair of the Pawtuxet Valley Agricultural Association. The knee buckles worn by Dr. Fiske are now in the Nathaniel Greene homestead, Anthony, R. I. (donated by his great granddaughter, Miss Kate F. Simmons).

Dr. Fiske was fortunate in money matters and liked a good mortgage. "Some farmers were debating the all-important matter of whether the old lady's apron in the moon was or was not a bit of good land and when the debate waxed warm, they decided to leave it this way—to search the land record and if any there was then Dr. Fiske must needs have a mortgage of it!"

"Among Dr. Fiske's private papers is a receipt from Sam Stone of $2,000, for a five year sorrel horse, purchased August 15th, 1780, showing the great value of the horses, or the small value of the currency of that day, which was probably about $60.00 in silver."

"One hundred and four years have already passed away since the settlement of the farm whereon I now live, then a dreary wild, was commenced by my Grandfather, Benjamin Fiske, aided by my Father, John Fiske, at the age of thirteen years, and for three generations has remained in our family. The recent and deeply lamented loss of my only Son, Philip M. Fiske, late of said Scituate, deceased, for whom I designed this Estate, induces me to devise it to my eldest grandson, Caleb Fiske Rea, for life with limitations over in hope it may continue a family estate for generations, yet unborn. I do therefore give and devise said farm with the house and other buildings thereon, containing by estimation Two hundred and seventy acres of land, to the said Caleb F. Rea for and during the term of his natural life, and also my Cranston meadow (so called) situate on the northerly side of the highway leading from Providence to Plainfield and passing directly by my dwelling house, containing eighteen acres, more or less, being land I purchased of William and Thomas Field of Cranston."

The description of this farm was taken from Dr. Fiske's will. The house burned several years ago. All that remains is the foundation, large chimney and stone steps which led to the cellar. The house was approached from the road by stone stepping stones and a large stone step still marks the location of the front entrance.

The small graveyard stands in the lot back of the house and contains the headstones of Dr. Fiske, his wife and children and his father and mother. It is surrounded by an iron fence, which has broken down in places. Many of the stones are moss grown and all need resetting.

The graves of Dr. Caleb and his father, John, are marked with a soldier's flag.

Across the road from the ruins of his home stand several barns and outbuildings, which look as if they dated back to colonial days.

Evidently Dr. Fiske gave thought to his personal appearance, for the inventory of his estate included 27 pairs of pantaloons and small clothes; 13 vests; 8 coats; a leather jacket; 1 Camblet great coat; 1 broadcloth great coat; 5 hats; 6 pair linen stockings; 6 linen shirts; 13 pair of mittens and gloves; 5 pair of shoes; 6 flag handkerchiefs, and many more articles of wearing apparel too numerous to mention.

He possessed a large and a small microscope; numerous medicines in bottles contained in drawers and a chest; saddle bags; tooth draws, and other medical instruments and a wheel chair. His home and office were well furnished, all articles being made of the finest mahogany. I have in my office an arm chair with rush bottom, and a sofa, which were used by him in his office.

His farm was well stocked with horses, cows, heifers, calves, one yoke of oxen and a bull. He also had a swarm of bees.

The photographs contained in this paper were made from personal effects and furniture used by Dr. Fiske.

Dr. Fiske's investments were chiefly in bank stocks and notes. At his death he left an estate valued at upwards of $40,000.

Dr. Fiske gave to two trusted women employees in the family $100 each and to several others $30 each, and to each of his heirs $50 each, all to be paid at the end of one year after his demise.

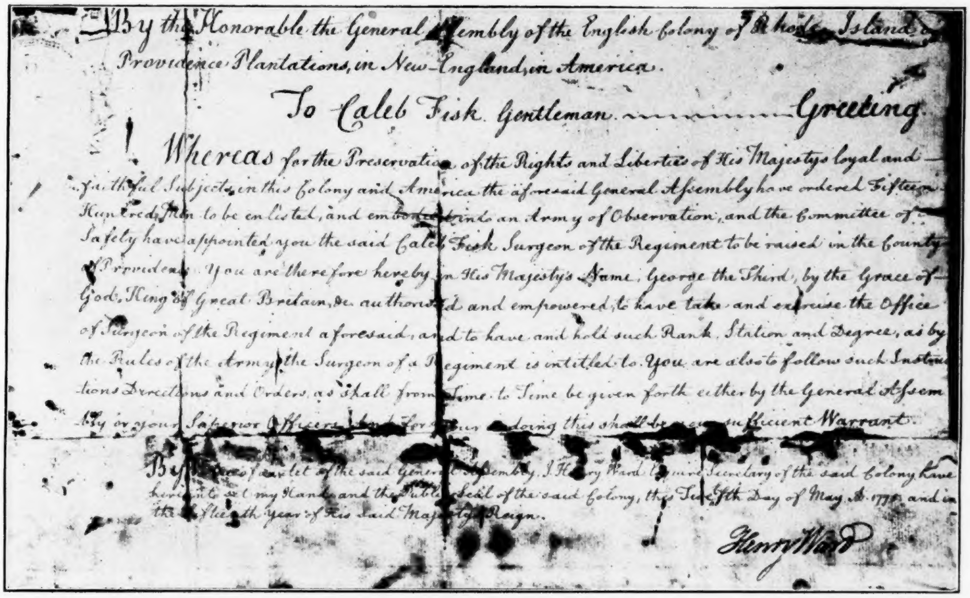
His property, personal and real, was given in trust for his grandson, Caleb Fiske Rea, his daughter and other grandchildren and their children.

I am indebted to the following persons who have made it possible for me to compile this rambling sketch, for furnishing me with historical data and personal property which belonged to Dr. Caleb Fiske:

Miss Abby E. Fiske, Mr. Richard Fiske, Miss Kate F. Simmons, descendants of Dr. Fiske.

Miss Grace E. Dickerman, librarian, the Rhode Island Medical Library.

The following articles were some of the personal belongings of Dr. Caleb Fiske

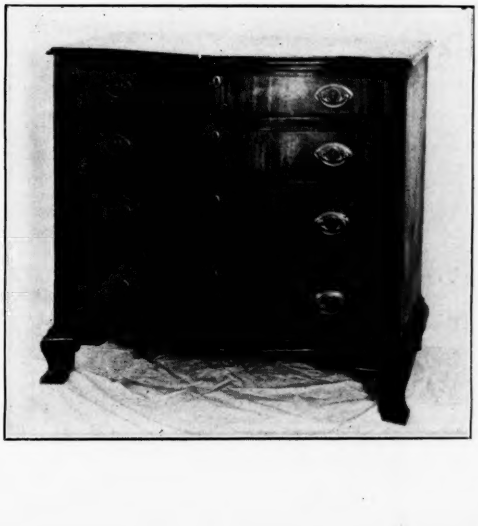

By the Honorable the General Assembly of the English Colony of Rhode Island and Providence Plantations, in New-England, in America.

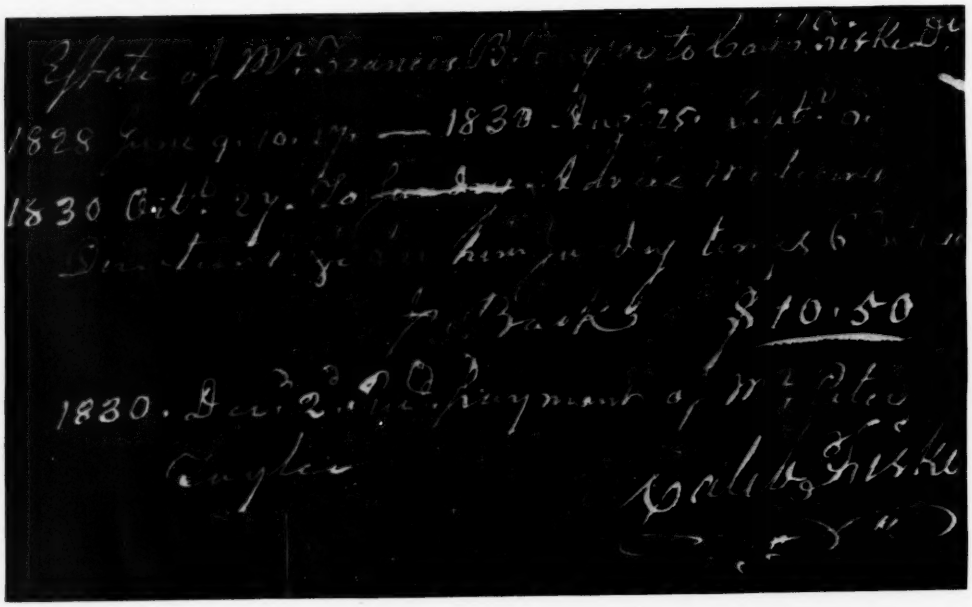
To Caleb Fisk Gentleman .... Greeting.

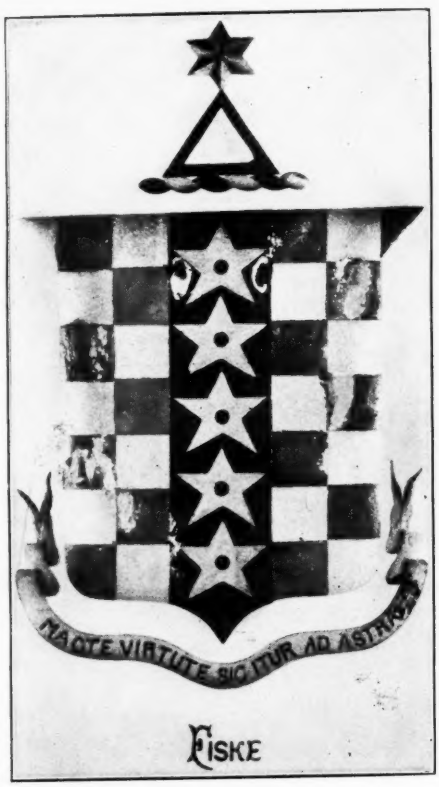
Whereas for the Preservation of the Rights and Liberties of His Majesty's loyal and faithful Subjects in this Colony and America the aforesaid General Assembly have ordered Fifteen Hundred Men to be enlisted, and embodied into an Army of Observation, and the Committee of Safety have appointed you the said Caleb Fisk Surgeon of the Regiment to be raised in the County of Providence. You are therefore hereby in His Majesty's Name, George the Third, by the Grace of God, King of Great Britain, &c. authorised and empowered, to have take and exercise the Office of Surgeon of the Regiment aforesaid, and to have and hold such Rank, Station and Degree, as by the Rules of the Army the Surgeon of a Regiment is intitled to. You are also to follow such Instructions Directions and Orders, as shall from Time to Time be given forth either by the General Assembly or your Superior Officers. And for your so doing this shall be your sufficient Warrant.

By [illegible] of the said General Assembly, I Henry Ward [illegible] Secretary of the said Colony have hereunto set my Hand and the Public Seal of the said Colony, this [illegible] Day of May A. 1775 and in the fifteenth Year of His said Majesty's Reign.

Henry Ward



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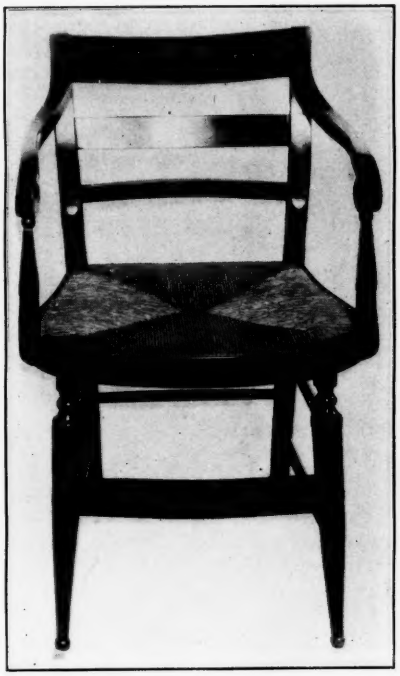

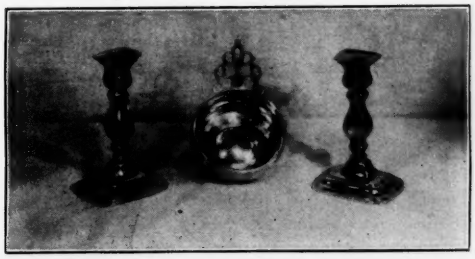

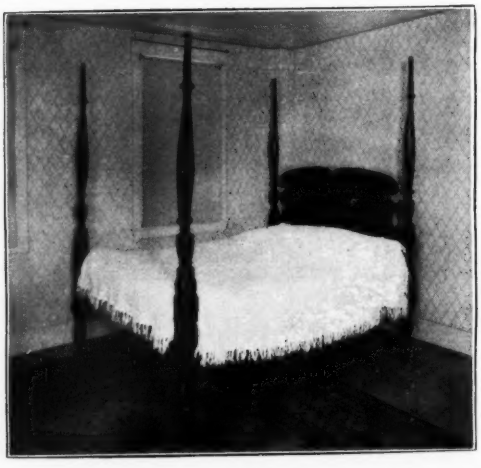

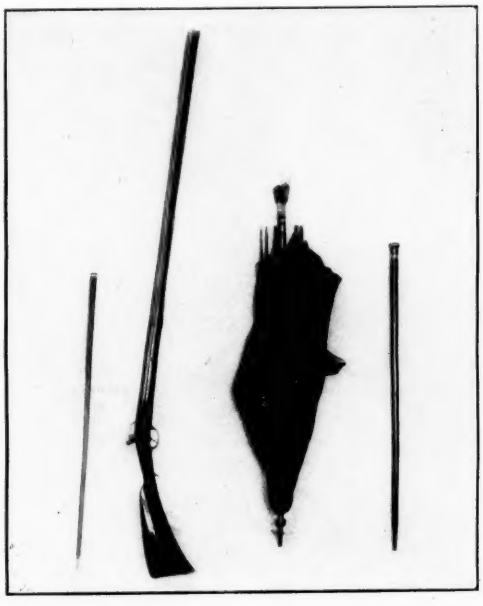

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R. I. Ophthalmological and Otological Society—2d Thursday—October, December, February, April and Annual at call of President. Dr. F. W. Dimmitt, President; Dr. N. A. Bolotow, Secretary-Treasurer.

The R. I. Medico-Legal Society—Last Thursday—January, April, June and October. Dr. Fenwick G. Taggart, President; Dr. Jacob S. Kelley, Secretary-Treasurer.

EDITORIALS

FIRST AID

The plan for the better understanding and treatment of fractures offered for the consideration and endorsement of the Rhode Island Medical Society is worthy of thought and study. It appears that eminent specialists in the treatment of emergency and first aid treatment of fractures of the long bones do not feel that such treatment and the immediate transportation of the injured is not competent and that the latest and best methods of treatment are being used. There is in this claim what can be construed only as an indictment of the medical profession and of the training in the medical schools. It may be said that any physician who is not capable of rendering first aid in an automobile accident is not fit to practice medicine, and that any medical school which does not prepare its men for such simple emergency work does not deserve to be upon the list of accredited institutions of surgical learning.

It is planned to send two recent graduates to district medical societies to instruct the members in these latest methods. These men are to receive the beggarly sum of $6,000 each per annum and are to

be equipped with X-ray films, exhibition boxes, movie outfits and the apparatus necessary to give lectures on first aid. There is probably no careful physician who would not be interested and instructed by such a demonstration. These young sirs are to be given serviceable automobiles and are to devote their entire time to this work.

Now in the first place it is not generally known that the medical profession is so remiss in its abilities, and we fall back on our original claim that there is something wrong if this is so—which we very much doubt. It must be obvious that a service such as briefly described here would cost a great deal of money and it is equally certain that it would be a very good thing and one from which nothing but benefit would result. But can it be claimed that the need is so urgent? Are there not other needs as great or greater? and is the profession as inefficient as the proponents of this elaborate and highly meritorious scheme fear? Earnestness and zeal are two of the most valuable qualities of any character. The conscientious welfare worker can think of a number of things which should be done for the betterment of home, food and living conditions. The economist will offer numerous suggestions regarding improvement in home and social economics, the cancer "research" man needs money, apparatus and what not, the eye specialist has sundry needs to be satisfied regarding the prevention of blindness, the ear man ditto in regard to deafness, and in fact each specialist has various plans which if followed would undoubtedly fill needs and improve the several conditions and situations. The writer would be the last to place any impediment in the way of medical progress, or to place aside lightly any such plan as this; but it does seem that we need more evidence to prove the real need and to substantiate the claim that the profession at large is incapable in the first aid treatment of emergencies. So this communication comes in the nature of a surprise; it is thought provoking and worthy of intensive study. The young medic who can enter a position assured with $6,000 cash money the "first year" is to be congratulated and one predicts that the competition will be keen and well attended.

PRINCIPLES OF MEDICAL ETHICS

In the "Principles of Medical Ethics," as published by the American Medical Association, one finds in Article II, Section 1, the simple statement, "Experience teaches that it is unwise for a Physician to treat a member of his family or himself."

Public opinion evidently from observation of the conduct of physicians has gotten the idea that it is illegal to treat members of one's family. Such is, of course, not the case, but no reputable member of our profession would or should undertake such a responsibility, yet, in certain quarters, one meets with glaring examples of this error in ethics.

For a State so well supplied with capable specialists as ours, to operate on one's own child "because there is no other good surgeon in town" seems to be a frank confession of puerile egotism, as well as such poor judgment as to reflect upon the surgeon's general prudence in all cases. To remove the tonsils or even as simple a job as to circumcise one's own son is not a matter of admirable courage but rather of immature common sense.

True, one can readily conceive of emergencies when the proximity of a familial medical man might save even life, let alone temporary relief of suffering and first aid. But is it not timely to remind ourselves that to deliberately undertake a not too emergent operation, or the care of a definite illness in the family, is bad judgment, bad ethics, and bad manners? Our profession is notoriously generous in the care of a colleague or his family and one may well consider it an honor to be asked to do so.

The old-time saying, "He who treats himself has a fool for a patient," may well be modified to read, "He who chooses his medical relative to treat him has selected an incompetent advisor." Affection and sentiment have a profound influence in this world of ours, but they have no place in scientific medical judgment.

SOCIETIES

The Rhode Island Medical Society

Council

Sept. 1, 1932.

A meeting of the Council was held following the general session of the Society, held at Warwick Country Club, Sept. 1, 1932, and on motion by Dr. Christie, duly seconded, the Council approved the resolution adopted by the general session approving of the proposition of the Fracture Committee of the New England Surgical Society with reference to post-graduate instructions in first aid, and transportation of patients with fractures of the long bones, and approved that the Rhode Island Medical Society appropriate $50.00 for the expenses of this purpose.

Adjourned.

Respectfully submitted,

J. W. Leech, *Sec'y*

House of Delegates

Sept. 1, 1932.

Immediately following the general session of the Rhode Island Medical Society held Thursday, Sept. 1, 1932, at the Warwick Country Club, the House of Delegates held a short meeting, and it approved upon motion made by Dr. Christie and duly seconded, the resolution of the general session approving

the proposal of the Fracture Committee of the New England Surgical Society to carry instructions in the first aid and transportation of patients with fractures of the long bones of the extremities. Also, the resolution of the general session approving the protest of the Newport County Medical Society against the free treatment and free hospitalization for non-service disabilities of veterans and their families was approved.

Adjourned.

Respectfully submitted,

J. W. LEECH, *Sec'y*

The regular quarterly meeting of the Rhode Island Medical Society was held Thursday, Sept. 1, 1932, at 4 P. M. at the Warwick Country Club with the President, Dr. N. D. Harvey, presiding.

Dr. Chas. L. Scudder, former Assistant Professor of Clinical Surgery of Harvard University, Chairman of the Fracture Committee of the New England Surgical Society, explained a plan of carrying to all the physicians of New England by means of demonstrations the first aid care and transportation of patients with fractures of the long bones of the extremities. The exact details of the plan have not been worked out, but the Committee expects to employ two recent graduates who will give their full time to these demonstrations before State and District Society meetings, and other groups of physicians as may seem feasible. The operation of the plan will be under the supervision of the above Committee and the New England Medical Council, which is made up of the Presidents and Secretaries and other members of all the New England State Medical Societies. In order to carry out the plans of the Fracture Committee, funds would be necessary, and it is the Committee's hope to present the approval and such appropriated funds as the State Societies feel able to make in an appeal to some established fund, which will bear the brunt of the financial burden incident to financing the plan. The matter was thoroughly discussed by the general session, and it was voted that the Rhode Island Medical Society approve the proposition of the Fracture Committee of the New England Surgical Society, and refer their action to the Council with the request for approval, and the appropriation of such funds as the Council deemed fit.

Dr. Norman MacLeod on behalf of the Newport Medical Society presented the following minute:

"The Newport County Medical Society is convinced that the privileges extended to veterans and their families for free treatment and free hospitalization for non-service disabilities works a hardship on the tax-paying public, the medical profession and the private hospitals. It is conceded that everything should be done for those who suffered disabilities in line of service. The Newport County Medical Society asks the Rhode Island Medical Society to join in urging the senators and representatives to stop any further privileges involving free medical service and, if possible, to rescind the present privileges that provide free medical service for non-service disabilities."

It was moved and seconded that the Rhode Island Medical Society concur in the contention of the Newport County Medical Society with reference to the free treatment and free hospitalization for non-service disabilities of veterans and veterans' families. It was so voted.

Program

First paper, "Dystocia due to Contraction Ring," Dr. Frank S. Hale, Providence. Discussion by Drs. Partridge, Appleton and Higgins.

Dr. Barton Cooke Hirst, Emeritus Professor of Obstetrics, University of Pennsylvania, who was to give an address to the Society, was detained by professional duties and unable to attend.

Second paper, "Transurethral Prostatic Resection," Dr. J. Edwards Kerney, Providence. Discussion by Drs. Stone and Turner.

The meeting was adjourned, and was followed by a buffet lunch.

Respectfully submitted,

J. W. LEECH, *Sec'y*

HOSPITALS

ST. JOSEPH'S HOSPITAL

The May meeting of the Staff Association was held on May 10, 1932. From the medical service, Dr. D. Frank Gray presented a case of myelogenous leukemia, presenting typical and characteristic clinical and diagnostic features. This was discussed by Dr. William Hindle.

From the gynecological service, Dr. Fred A. Coughlin read a paper, entitled "Radium, Its Application and Indications, with Special Reference to Gynecology." It comprised an accurate historical account of its discovery, its subsequent development through several phases, and its mode of application, the indications for its use, and concluded with statistical references and review of results accomplished by its use especially in carcinoma of the cervix. This interesting paper was discussed by Drs. Gerber, Boyd, McGuirk, and A. W. Mahoney.

The Staff was tendered a complimentary banquet by the hospital, through the Reverend Mother Superior, on May 24, about 125 members being present.

An informal outing attended by nearly 100 members was held on June 15.

Announcement of the renewal of the series of Clinico-pathological Conferences conducted by the Interne Staff was made this month, the first of the new series taking place on September 16, and each Friday thereafter, at 12:30 P. M.

The New England Conference of the Catholic Hospital Association notice, herewith attached.

JOSEPH L. BELLIOTTI, M.D.,
Secretary

The New England Conference of the Catholic Hospital Association was held at St. Joseph's Hospital, Providence, Rhode Island, August 30, 31, September 1, 1932.

His Excellency, William A. Hickey, D.D., Bishop of Providence, presided at the opening session; in his address to the delegates he stressed the importance of charity in our hospitals.

Honorable James E. Dunne, Mayor of Providence, welcomed the delegates to the city of Providence, and discussed "The Importance of Hospitals and Health in a Community."

Interesting and instructive papers were read by the following doctors of the Staff:

"Our Interest in the Crippled," by Dr. Wm. Horan.

"Relations of the Attending Staff to the Intern," by Dr. Frank McEvoy.

"The Essentials of a Laboratory in a Modern Hospital," by Dr. Francis W. Constable.

During the convention the delegates visited the Rhode Island Hospital, also the Charles V. Chapin Hospital.

The Conference adjourned Sept. 1, 1932.

JOSEPH L. BELLIOTTI, M.D.,
Secretary

MISCELLANEOUS

PARENTERAL LIVER THERAPY IN TREATMENT OF PERNICIOUS ANEMIA

Maurice B. Strauss and William B. Castle, Boston (*Journal A. M. A.*), have been unable to detect any difference in effect on blood formation between intravenous and intramuscular injection. However, reactions accompanied by chill and fever occurred in about one-third of patients in relapse who received an initial intravenous injection, and in one patient with a history of natural allergy there occurred a severe non-fatal shock following the third intravenous injection at weekly intervals. This was the only alarming reaction among about 200 intravenous injections. However, since the intramuscular method did not produce a systemic reaction from any one of over 2,000 injections in more than 100 patients, the authors abandoned the intravenous route altogether, although occasionally one may prefer the intravenous administration of the material to the intramuscular when large doses must be given. The treatment of the average patient with pernicious anemia in relapse may be accomplished satisfactorily by the daily intramuscular injection of 2 cc. of liver extract. The extract is a simple water solution of liver extract No. 343 (N. N. R.), now brought without buffer to *p*H 7.4, filtered and preserved by the addition of tricresol. In an emergency, liver extract No. 343 derived from 100 Gm. of liver (the contents of one vial) may be dissolved in 20 cc. of warm water, filtered, boiled for five minutes and injected with the only disadvantage that the intramuscular injection is painful, whereas a similar amount of the properly neutralized extract may be injected at one time without excessive discomfort, and from 2 to 5 cc. will seldom cause any discomfort whatever. Extracts of greater purity have been repeatedly employed, and it has been found that further fractionation or removal of material results in a loss of potent material. Furthermore, if the dry extract (No. 343) derived from 100 Gm. of liver (about 4.5 Gm.) is dissolved in less than 20 cc. of water, there is reason to believe that the solution is not as effective, probably owing to the failure of all the potent material to enter solution. The question of a maintenance dose cannot be settled at this time. When, in addition to a blood normal in all respects, consideration of all aspects of a case reveals no remediable abnormality, the dose may in certain instances be reduced. In the usual case, two or three injections of 2 cc. of extract a week, or a single weekly injection of from 5 to 10 cc., presumably may suffice. However, in the resistant case much more than this amount will be needed. In each case the blood and general condition should be studied at frequent intervals to insure adequacy of treatment. It has been the authors' practice to continue daily injections of at least 2 cc. of extract in all cases with neurologic manifestations, irrespective of the fact that smaller amounts will maintain the blood at a normal level. How long this dose should be continued remains for the future to decide. The keynote of therapy should be always to give more than "just enough." Intramuscular liver therapy has been found of great benefit in "resistant" cases of pernicious anemia and in cases presenting symptoms due to spinal cord degeneration.

NEWS ITEM

Dr. Louisa Paine Tingley has been reappointed Councillor of the Massachusetts Medical Society for 1932 and 1933.

FISKE FUND PRIZE ESSAY, NO. LXVII

THE VALUE OF OCULAR SIGNS AND SYMPTOMS IN THE DIAGNOSIS OF GENERAL DISEASE

HARRY C. MESSINGER, M.D.

PROVIDENCE, R. I.

"Ex orbe lux"

THE Trustees of the Fiske Fund, at the Annual Meeting of the Rhode Island Medical Society held at Providence, June 2, 1932, announced that they had awarded a premium of two hundred dollars to Harry C. Messinger, M.D., of Providence, Rhode Island, for an Essay entitled "The Value of Ocular Signs and Symptoms in the Diagnosis of General Disease."

DR. HARRY L. BARNES, Wallum Lake, R. I.
DR. N. DARRELL HARVEY, Providence, R. I.
DR. CHARLES S. CHRISTIE, West Warwick, R. I.
Trustees

WILFRED PICKLES, M.D.
Secretary to the Trustees
184 Waterman Street, Providence, R. I.

THE VALUE OF OCULAR SIGNS AND SYMPTOMS IN THE DIAGNOSIS OF GENERAL DISEASE*

VERY few eye disorders are due to disease originating in the eye itself. In intraocular inflammations, muscle-palsies, affections of the retina and optic nerve, and often even in the case of lesions of the cornea and conjunctiva, the question of cause immediately leads away from the organ itself. A complete medical examination is routine procedure in all but the simpler cases in modern eye diagnosis.

In return, examination of the eye may be of help in the diagnosis of general disease, very rarely in the positive proof of the specific, etiological cause but frequently in the enlargement and better definition of the clinical picture in the individual case.

To illustrate this, after a consideration of some of the conditions which affect the manifestations of general diseases in the eye, clinical studies in groups of cases will be reviewed with especial regard to their diagnostic value.

Against injury from without, the eye is wonderfully protected. To attack by way of the blood stream it presents no special defense, and its reactions to blood-borne irritants, toxins, and infections, in nature not different from those in other parts of the body, are, because of the specialized structure of the tissues involved, characteristic to a great extent, of the reacting tissues rather than of the specific nature of the irritating, toxic, or infecting agent.

Most tissues of the eye have mesodermal and ectodermal elements and a supporting structure and blood supply. The eye may be thought of as a part of the brain especially differentiated for the function of sight. The retina has nerve cells with delicate end processes and axis cylinders, whose integrity and function are linked with those of a complicated system extending through a great part of the brain; the retina indicates remarkably the location of disorders of this system. Six of the twelve cranial nerves have such relationship to the eye that disturbance of their function may produce eye signs.

The eye may be considered an appendage of the skin, evolved as an organ of protection and containing, in the retina and uveal tract, pigment derived from the skin and having the same characteristics as skin-pigment wherever found.

The circulation of blood and body fluids in this small organ is remarkable and important in connection with our subject. In the smaller vessels of the conjunctiva, by means of the corneal microscope, individual blood-cells may be seen tumbling along through narrow tubes just large enough to let them pass one by one. With the ophthalmoscope the blood stream in the retinal arteries and veins may be inspected, magnified sixteen diameters. Just beneath, most of its details covered by the more or less opaque pigment layer of the retina, is a vascular bed, a plexus, almost a lake of blood, the choroid, a circulation independent of the visible retinal vessels.

The eye, moreover, has a circulation of its own, necessary because of the need of transparent, bloodless media for the transmission of unobstructed light through the cornea, the anterior chamber, the lens, and the posterior chamber with its vitreous body of colloid-gel bathed in the same fluid which fills the anterior chamber. The complicated physiochemical actions and reactions by which the structures of the eye are kept in anatomic integrity and functional equilibrium, and their processes of waste and repair maintained, are not well understood. An important agency is the intraocular fluid which is a dialyzate from the blood-serum through the endothelium coating the ciliary processes. The physical and chemical properties of this fluid have been studied. It contains all the constituents of the blood-serum but in different proportions; it probably is a mass-collection of the tissue-fluid which also fills the tissue spaces in the uvea, cornea, vitreous-body, retina, and even the sclera. It is essentially the salt water which bathes every living cell and which is the medium in which we really live. It resembles closely in its chemical, ionic and hydrodynamic properties the cerebro-spinal fluid. Its dynamic and physical properties can be

**In view of the expressed desire of the Trustees that Dissertations submitted in the Fiske Fund competition represent the personal work of the author, I have taken the liberty of limiting my consideration of the subject to those phases in which I have made a special study during the past few years. My essay, accordingly, will deal chiefly with the ophthalmoscopic appearances observed in patients having diabetes, diseases of the blood, or hypertension.*
H. C. M.

studied clinically in only a limited way. If it could be withdrawn and examined microscopically and chemically as is possible in the case of the spinal fluid, it would help us greatly.

Diabetes of the Eye, 100 Consecutive Cases

As regards eye complications in diabetes, there seems to be a natural division of the cases into two groups, the young with severe diabetes and rare eye disorders, and the older (40 or more) with milder diabetes and frequent eye complications. There are, of course, some individuals between 40 and 50 years of age whose diabetes is severe and occasionally cases under 40 who have a diabetes and, perhaps, constitutional traits or tissue changes characteristic of the aged. In a large series such atypical cases would undoubtedly appear, but this division is of value, especially with respect to eye signs.

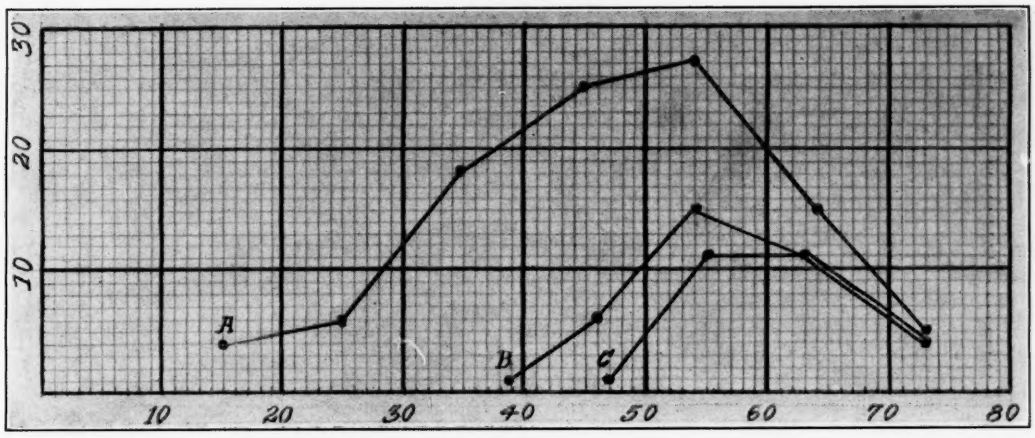
The young diabetics have good blood vessels, but the aged show, as a rule, arteriosclerosis and often have hypertension associated with changes in the small, precapillary endarteries—arteriolar sclerosis. So that, although typically the disturbance of metabolism is more marked in the young, eye complications, particularly those of vascular origin, are very infrequent. In this series of 100 cases, in 8 cases under 30 years of age, where the known duration of the disease averaged 4 years and 5 months, objective evidence of eye disease was lacking, although one patient 21 years old was known to have had diabetes 15 years and required 60 units of insulin daily and closely supervised diets to keep his diabetes under proper control. Another patient, 31 years old, a known diabetic for 5 years, who had twice been in coma but had successfully gone through two pregnancies in that time, and who required 40 units of insulin and regulated diet to control her metabolism, showed no eye trouble. In contrast with this group are the five patients in the eighth decade of life, four of whom showed cataractous changes in the lenses and four retinal arteriosclerosis (in one the retinae could not be examined because of cataract in one eye and vitreous opacities in the other).

Many of our cases complained that at some time, usually during the period when they were first put under insulin therapy, they had disturbances of vision which later cleared up. Such cases represent undoubtedly either changes in the refractive power of the lens or paresis of accommodation, a condition not rare in diabetics untreated or ineffectively treated. Such patients may, because of poor vision, consult an oculist before they realize the need of the services of an internist. Paresis of accommodation also is common in the first weeks of insulin treatment, while the neglected and abnormal metabolism is being changed materially in a few days, by diet and the administration of insulin. This weakness of accommodation is not serious; it disappears as the new metabolic condition is stabilized, usually in from two to four weeks. The factors involved in faulty metabolism and metabolism regulation are not simply sugar and acidosis or acidemia; water and salts are important. The severely afflicted diabetic has a general dehydration, a lack of the salt water which fills the intercellular spaces of the body, and the injection of insulin causes, per contra, a retention of salt water, and may produce an insulin-edema. Besides this retention there are changes in the ionic concentration of the body-fluids; a transmineralization takes place, the particular consideration of which is out of place here. Accommodation-paresis may depend on these alterations from the normal, either of sugar and acid products or of the water-and-salt content of the organism and its parts.

In the changes of refraction which occur in diabetics and which often cause the patient to consult an oculist, it is evident that the water-and-salt factor is important etiologically. An acute myopia, presumably due to swelling of the lens, if not due to an increase in refractive index, cannot be explained by the changes in the sugar and acetone bodies but can be understood on the basis of salt-water metabolism changes; such an acute myopia occurs in a diabetic in bad general condition. It is difficult to say whether a hyperopia appearing during treatment is due to decreased lens-refraction or to accommodation-paresis; perhaps both are involved. A change of refraction of as much as 13 diopters (from plus 4 to minus 9) has been cited (Umber: Text book of Nutrition and Metabolism). Most of these refractive changes occur in severely afflicted diabetics, but under modern treatment such cases may not always show a marked glycosuria.

In diabetics in coma, or where coma seems impending, the eye-ball is sometimes very soft. This is an evidence of the extreme dehydration of the body, a severe type of metabolic disturbance. Only in diabetics is the interesting condition, lipemia retinalis, seen. There the retinal vessels are orange-yellow in color, due to the high fat-content of the

100 DIABETICS



A Total Number of Cases Each Decade
B " " " " Retinal Arteriosclerosis
C " " " " Lens Changes

A				B			C		
Decade	*Cases*	*Age*	*Duration*	*Retinal Arteriosclerosis*			*Cataract*		
1st	0	0	0				1	47 Yrs.	
2nd	4	15 Yrs.	4 Yrs. 4 Mos.	1	39 Yrs.		11	55 Yrs.	Av. Age
3rd	6	25 Yrs. 6 Mos.	6 Yrs. 2 Mos.	6	46 Yrs.		11	63 Yrs.	
4th	18	34 Yrs. 7 Mos.	2 Yrs. 7 Mos.	15	54 Yrs.	Av. Age	4	73 Yrs.	
5th	25	44 Yrs. 8 Mos.	2 Yrs. 9 Mos.	11	63 Yrs.				
6th	27	54 Yrs. 3 Mos.	3 Yrs. 7 Mos.	4	73 Yrs.				
7th	15	64 Yrs.	7 Yrs.						
8th	5	73 Yrs.	4 Yrs.						

blood. The cases in this series were all ambulatory, and in them one would not expect to see such extreme manifestations.

Not many years ago very good observers (Naunyn: Text book) considered that in an eye-muscle disorder appearing suddenly one must think first of diabetes, but more recent reports of series and clinicians asked about this point, agree that it is not common; we saw none in these cases. When it does occur as a result of diabetes, more commonly in the sixth nerve, it clears usually in one to two weeks. I saw one case of papillo-macular-bundle atrophy, evidenced by temporal pallor of the discs and large, absolute, central scotomata; but the blood Wasserman was 4 plus and arsphenamine had been given, casting much doubt on diabetes as the cause.

The diabetic is especially liable to inflammatory and purulent infections, or at least makes poor resistance to them. We had one case blind in one eye from post-operative inflammation, two cases of chronic dacryocystitis and one of chronic iridocyclitis. With modern dietetic and insulin treatment much improvement has come about; this is certainly true of the inflammations formerly so much dreaded after operations on the eye. In a diabetic suffering from intraocular inflammation, search should be made for other possible etiological factors such as syphilis, tuberculosis and foci of infection: a diagnosis of diabetic iritis, for instance, is not satisfactory.

Diabetic retinitis, so called, I saw in six cases. The youngest patient exhibiting it was 39 years old. It does not occur in young diabetics and it is not common in the old with severe diabetes. There is little doubt that it is dependent mainly on blood-vessel changes of a sclerotic nature which appear with increasing frequency as age advances. Nevertheless the typical form, with white, focal spots in the region from the papilla to the macula, is rarely seen except in diabetics, and it must be freely admitted that the metabolic disorder has some effect in determining the type of retinitis. In retinal arteriosclerosis hemorrhages are seen usually in well advanced cases, but, apparently, in diabetics hemorrhages and exudates (that is to say "retinitis") occur when, to ophthalmoscopic appearances the vessels show only moderate alterations from

normal. Diabetics are said to be especially prone to cerebral hemorrhage.

Retinal arteriosclerosis was present by definite signs such as narrowing and pallor of the arteries, corkscrew dilatations of the small venules about the macular region, Gunn's pressure sign at arterio-venous crossings, etc. In 37 of our cases, 14 out of 26 males and 23 out of 74 females: 1 out of 18 cases in the fourth decade; 6 out of 25 in the fifth; 15 out of 27 in the sixth; 11 out of 15 in the seventh; and 4 out of 5 in the eighth, (the fifth case had a mature senile cataract in one eye and asteriod hyalitis in the other, so that a view of the fundi was impossible).

In the older diabetics cataract is undoubtedly more common, appears at an earlier age, and develops more rapidly than in non-diabetics. The rare double-sided cataract of young diabetics has been considered as specifically due to diabetes. It is idle to speculate on its exact pathogenesis; perhaps both it and the severe juvenile diabetes which it accompanies represent manifestations of a constitutional inferiority.

The cataracts in our cases all seemed to be senile. One of our cases had a cataract removed when she was 52 and diabetes appeared at 56.

Senility and vascular degeneration are important factors in the eye-complications of the older diabetics.

In intraocular inflammations or in disturbances of the neural mechanisms connected with the eye, other etiological factors should be sought as thoroughly as if the diabetes did not exist.

The eye tells little of the diabetes but much of the diabetic.

Arterial Hypertension and the Retina

In the presence of persistent hypertension the retina and its vessels may show:

1. No observable change from normal,
2. Evidence of retinal arterio-sclerosis,
3. Retinitis, or neuro-retinitis

To arrive at some basis for interpretation of these different findings we must first have a working, theoretical explanation of their meaning. In the absence of any knowledge of the cause of hypertension, our theory may be in part false, but nevertheless useful.

Essential hypertension, we assume, begins with a contraction of the invisible, pre-capillary arterioles; for a long or short time these may be contracted and the visible vessels, even the smallest, show no change. In such cases the retina and the retinal vessels show ophthalmoscopically no alteration from the normal. This normal appearance of the retina in the presence of high blood pressure we have also seen in youth, persisting for five years, where there was no doubt of the clinical diagnosis and where the blood chemistry and urinary findings confirmed the presence of a chronic nephritis. At this point it may be noted that nephritis, even to the point of death in uremia, may occur with normal findings in the retina.

In the absence of demonstrable nephritis, hypertension, with retinal vessels normal in appearance, may persist for years. Many times histological examination of such retinae shows arteriosclerotic changes in the small vessels. As far as it goes then, such normal retinal appearance speaks for a better prognosis than we have in group two, where we have evidence of retinal arteriosclerosis.

These have been described by many observers. My theory (I claim no priority, but here will try to extend it to cover all cases exhibiting high arterial pressure) is, in this connection, that, after a longer or shorter time, not only invisible arterioles but gradually the arteries of greater size become smaller in caliber and their walls thickened by increase in the adventitia and thickening and degeneration of the media, ischemia of the retina gradually resulting.

The most important signs of these changes are: narrowing of the arterial stream particularly in the smaller vessels as seen by the ophthalmoscope; "pressure sign (Gunn's)," apparent narrowing of the vein where it is crossed by the artery, sometimes to such an extent that the venous stream appears to end near the artery and begin again on the other side; and dilatation of the small vessels, mostly tiny venules in the peri-macular region which, besides increased visibility, often show tortuosities (corkscrew-tortuosities). Other less constant signs are: increase of the white reflex on the arteries; inequalities and irregularities in the caliber of the arteries; white accompanying streaks along the arteries and veins; and, later in the disease, retinitis—blurring of the discs, small hemorrhages in the vessel layers of the retina and finally macular degeneration, larger hemorrhages and exudates.

These changes have been described about in the sequence in which they develop. We need observations of cases of hypertension over periods of years and decades with correlation of the general clinical

Arteriosclerosis of a retinal artery; hyalinization of the media; woman 72 years of age.

and retinal findings. It is possible that retinal arteriosclerosis may occur in the absence of hypertension, but where the signs such as relative narrowing of the arteries, corkscrew tortuosities of the small vessels, and the pressure-sign of Gunn are prominent, it may be assumed that hypertension exists or has existed. As these are the only small vessels visible to the eye it is possible to gauge with considerable accuracy the extent to which the individual's arteries of this type in other organs have been affected by the arteriosclerosis. In the vast group called "hypertensive disease" the examination of the retinal vessels should not be neglected; it can be made a part of the general physical examination.

In a study of 200 ambulatory patients presenting hypertension I found arteriosclerotic changes in the retinal vessels in 151, about 75%. There was evidence of previous cerebral hemorrhage or thrombosis in 21, about 10%; urine of low specific gravity in the majority; evidence of cardiac hypertrophy in 70 cases.

Death in hypertension usually results from heart disease, kidney disease or cerebral accident. Hypertension per se may exist for years without symptoms, but an examination of the retinal vessels is often of great value in prognosis; if they are normal in appearance that is favorable (though it does not rule out the possibility of cerebral hemorrhage); the presence and degree of development of changes suggest similar changes in the vessels of the brain, the kidneys and the pancreas; the presence of such changes where there is clinically a myocarditis, may indicate that the myocardium is weak from long continued effort to maintain a high blood pressure even though the pressure at the moment is low. In the presence of a cerebral lesion it may help the diagnosis of a vascular cause.

Retinitis, or neuro-retinitis, associated with hypertension is found in advanced cases of retinal arteriosclerosis; in the vascular type of chronic nephritis, sometimes with increased intra-cranial pressure (with or without kidney involvement); and in the toxemic states of late pregnancy.

Examination shows edema of the retina and swelling of the optic disc, areas of transudation and retinal hemorrhage, sometimes localized areas of retinal edema and, about the macula, the so-called "star-figure"—radiating, yellowish-white areas of retinal degeneration, arranged much as the spokes of a wheel or a part of a wheel, about the fovea as a hub. In some cases the papilledema is the most important sign (a differential diagnosis of brain tumor is sometimes difficult). The thorough histopathological study of such a case under most fortunate circumstances by Igershemier (Schnervkrankung bei maligner Sklerosie Z. F. Augenh. V. 69 N. 1-3 September 1929) demonstrates that a purely vascular cause may produce the picture of choked discs, retinal edema and the star-figure about the macule. Commonly however the edema of the discs is less prominent than the retinal changes.

The retinal changes in arteriosclerosis of the retinal vessels come on slowly as a rule; they are probably due to the reduced supply of blood through the affected vessels; at first scattered and a few, they may finally produce the complete picture of a hypertensive retinitis.

The retinitis of kidney disease has been known and its prognostic value recognized for 70 years. It was called albuminuric retinitis. In the light of our present knowledge this term is poor. Retinitis is not seen in the kidney diseases where the urine contains the greatest amounts of albumin, the nephroses. It is seen in that class of cases where small vessels afferent to the glomeruli are particularly involved, the vascular type of nephritis, where often there is very little albumin in the urine. Also misleading was the term "azotemic retinitis." In kidney disease where nitrogen-retention products are either high or low, retinitis may or may not be present. Where retinitis does occur in nephritis there is arterial

hypertension; if to hypertension another factor must be added it is as yet X, the unknown quantity.

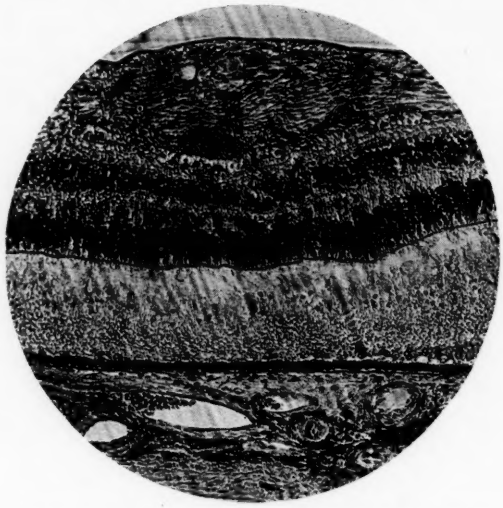
In "malignant hypertension" we have this neuro-retinitis in all its qualities; the papilledema is often great, the retinal hemorrhages are frequently large, but none of the characteristic signs are missing in most cases. The increased intracranial pressure may be due to an interstitial edema of the brain. Involvement of the kidneys may occur after the eye and cerebral symptoms have been present for some time or may never develop. Hypertension is great, not relieved materially by treatment and characterized by high diastolic readings. Here we have hypertension, retinitis and hypertensio-cerebri, the ultimate cause or the cause of the hypertension being absolutely unknown. The prognosis in malignant hypertension is grave; cerebral decompression is of almost no palliative help; dehydration of cerebral tissue by intravenous or alimentary routes of more value.

A study of the retina in cases of toxemia in the later months of pregnancy, 108 cases in all, 75 of which were diagnosed as eclampsia, and 33 as "pre-eclamptic" toxemia, gives us an opportunity to observe hypertensive retinitis, or better, neuro-retinitis, in all stages of its development, and, what we do not see in malignant hypertension with or without nephritis, its regression, sometimes to complete recovery. Many of these cases were seen in the pre-natal clinic when the eye grounds had a normal appearance; seen there or in the hospital wards as their signs and symptoms indicated an increasing general "toxemia"; the earliest changes noted in their retinae; and followed through the entire course of their disorder, some of them in the post-natal clinic as late as five months after discharge from the hospital wards.

Of 79 cases with systolic blood-pressure over 150 mm., 45 showed a retinitis of some degree; of the 29 cases with systolic blood pressure less than 150 mm., none showed retinitis. Of the 33 cases having convulsions and diagnosed as eclamptics, 24 had systolic blood pressure over 150 mm. and 11 of these showed retinitis.

The urine in these cases with retinitis varied from a low-gravity urine with a few casts or a few red cells and no casts, up to a concentrated, scanty urine with much albumin and many blood cells and casts.

Some degree of edema of the face, back or the extremities was seen in each of these 45 cases positive for retinitis. Since these records were made, one case of eclampsia has been observed with hypertension and retinitis but with no apparent edema under the skin.



Advanced case of hypertensive neuro-retinitis. There is a mass of scar tissue in the deep (uppermost) layers of the retina; between the two molecular (black-staining) layers are many hemorrhages (in this picture the blood corpuscles are black); the choroid and retina are separated by serum.

Hypertension was a constant sign and, as has already been said, retinitis was not present in any of the 29 cases without hypertension.

The retinitis in some cases developed very slowly (2 to 4 weeks), in others appeared suddenly and developed rapidly. Cases in the pre-natal clinic with rising blood pressure, edema or other signs indicating the beginning of a toxic state were examined for retinal condition and thereafter followed regularly. The first evidences of retinitis were slight blurring of the disc-margins and general haziness of the retinae, making the vessels appear slightly indistinct in outline. Gradually the haziness increased so that, in places, the vessels became very dim in outline or disappeared from view. At this stage, in some, oval or roughly oblong, sharply edged, white patches could be seen beneath the retinal vessel levels; these varied from 1/8 to 1/3 disc-size in area and were most commonly from 1 to 3 disc-diameters from the disc. They had an appearance suggesting small separations of the retinae. Whether or not these were seen, with increasing general signs of systemic disturbance then began to be seen hemorrhages into the retina, usually streaked and along the vessels

Papilledema in a case of "albuminuric retinitis"; the retina is displaced laterally and slightly folded; there is serum between the choroid and retina.

(especially veins) and, adjacent to veins, hazily outlined, pale areas, blurred, "cotton spots." At this stage the veins were large and dark, the arteries narrow; in many places the vessels indistinctly seen, sometimes dipping in and out of hazy retina or overlaid in part by masses of fuzzy, colorless structure, the disc margins obscured, even often to the appearance of a marked papilledema. Then about the macule appeared the star-figure we have already described. Vision at this stage was reduced, in the sickest patients to almost complete blindness.

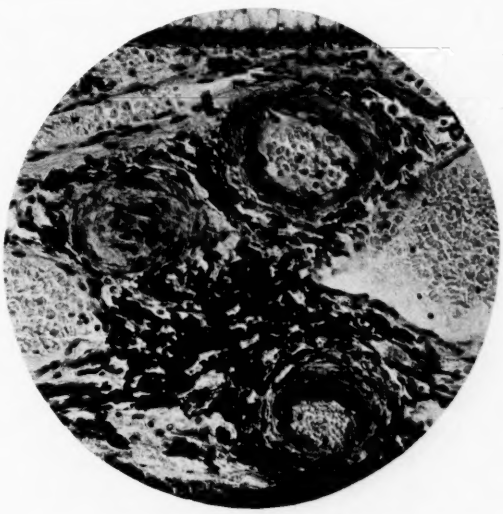
At any stage in this development of a full-blown retinitis, with the general improvement which usually set in with either spontaneous or forced emptying of the uterus, there was rapid regression of the retinitis, disappearance of the hemorrhages and transudates, subsidence of the blurring of the discs and, if it had developed, the star-figure was gradually replaced by an indefinite mottling of the macular area.

Case 1. (Patient of Dr. Ira H. Noyes):

P. F. A primapara, 34 years of age, had a complaint of blurred, poor vision without notable retinal signs; within 24 hours, however, the disc-margins became blurred, the retina hazy, and the vessel outlines slightly indistinct; at this point labor began and lasted only three hours; within another 24 hours vision had become practically normal and in two days the retina again had a normal look.

In eight cases characterized by sudden onset of the "toxic" symptoms, rapidly rising blood-pressure, so that from a normal or slightly supra-normal pressure the systolic pressure rose to 230-270 and the diastolic to 130-160, severe headache, increasing fogging of vision (in what might be called a fulminating type of case) it seemed characteristic that the discs and retinae became rapidly swollen and had a watery look. The vessels, both arteries and veins, seemed narrowed and straightened. Most of these cases did not go on to the stage with hemorrhages and the star-figure; as the pregnancy ended and the headache and high-blood pressure subsided and the vision returned, the swollen watery look rapidly left the nerve-ends and the retinae; at this stage a few hemorrhages and in some, a faint, incomplete star-figure appeared but persisted for only a few days.

Three times in this series retinal separation was observed. In one case it was about four times the size of the disc, temporally beyond and slightly below the macula. This was an emergency case, one of the eight fulminating cases we have just described. Twelve hours after delivery the separation had entirely disappeared; the other signs were those of retinal edema previously described and papilledema. The second case was one of particular interest.



Choroid in a case which clinically showed hypertensive neuro-retinitis; the arteries sclerotic, the lumen of one obliterated; the walls of the veins not altered; woman 23 years old.

Case 2. (Prov. Lying-in Hosp., 11696):

A primipara, 25 years old, at term; within a week she had been examined by her family doctor who found no threatening signs, the blood-pressure S. 150 D. 90. On the morning of the day of her admission to the hospital she had a severe headache; at 5 in the afternoon she complained of blurred vision; at 6 she was seized with a clonic convulsion; after which she said she could see nothing; she became rapidly stuporous, spastic and resistant, and at 7:30 p. m. had a second convulsion. When examined at 8 p. m. a separation from above, estimated to involve one-third of the entire retina, was present; the separated portion of the retina showed general edema; the disc was obscured by it. At this time the blood pressure was S. 210, D. 130; the face and extremities were puffy; a few ounces of concentrated urine with much albumen, many red cells and casts, were obtained by catheter. Sixteen hours after the forced delivery the separation had disappeared and and in four days the general condition had improved; the retinal edema had gone and vision was normal.

The other case was in a patient, also a primipara, who had for three weeks shown hypertension with no changes in the retinae except slight retinal edema; suddenly she developed severe headache, vomiting, rise in blood-pressure and, in a few hours, edema and marked diminution in urinary secretion. Examination revealed a separation of the retina from above. As in the two previous cases there was quick recovery after delivery. One of my colleagues observed a similar case in which there was almost complete separation in both eyes with "re-attachment" within 48 hours after delivery.

If the retinal signs I have described and called hypertensive retinitis form a clinical entity, separations such as I have related in discussing the "toxic" states of late pregnancy should sometimes occur in malignant hypertension with or without nephritis. The following case is an example of separation of the retina in neuro-retinitis nephritica.

Case 3. (Patient of Dr. Joseph C. O'Connell):

A ten-year-old boy, complaining of sudden loss of vision in his right eye, had suffered for three months with hematuria and vomiting. The urine contained, besides much blood, hyaline and granular casts. The blood showed Hgb. 60%; red count 3,500,000; blood urea 78 mgm.%; creatinin 2.4 mgm.%; B.P., S. 180 D. 110. He complained of poor sight, and examination showed in the right eye a separation of the retina involving the lower two-thirds of the retina; in the left eye, neuro-retinitis with marked general retinal edema and a few scattered, streaked hemorrhages. Death followed in less than 48 hours after the complaint of loss of sight.

For this retinal picture which has been observed associated with increased intracranial pressure with or without evidence of vascular nephritis and which is the characteristic retinal appearance in the toxemic states of the late months of pregnancy, the most appropriate name seems to be "hypertensive neuro-retinitis," expressing the three principal signs, arterial hypertension, papilledema and retinal changes (ischemic reaction; stasis, transudation, hemorrhage and degeneration).

According to our "working theory" there is here a contraction of the pre-capillary arterioles, in the nature of unremitting and strong spasm. In the early stages of essential hypertension there is a similar spasm of the vessel walls, but less intense and less constant. In this neuro-retinitis there results an ischemic reaction in the retina; in retinal arteriolar-sclerosis and subsequent arteriosclerosis the diminution in nutrition by the way of the blood stream is very much more gradual, a matter of years, or decades, instead of hours or days.

A constant accompaniment of this neuro-retinal edema is some degree of interstitial edema of the brain. Where the cerebral changes are relatively great we have "malignant hypertension." Where they are relatively slight compared to changes in the small vessels of the kidneys, we have the same eye-picture under the old title "albuminuric retinitis." There is some common factor in the pathology of vascular nephritis, malignant hypertension and eclampsism; and there is the possibility that in essential hypertension there is a similar factor, less severe and pronounced, not catastrophic in its manifestation.

Aside from these rather theoretical values of retinal changes in hypertensive states there are the values in the individual case with hypertension. Blood pressure above the commonly accepted normal limits unsupported by any other clinical evidence is important but of relatively little diagnostic significance. The retinal changes we have described add much to the picture and their value increases with the development of the general clinical evidence.

The Retina in Anemias and Allied Disorders

The discussion in this chapter on the retina in anemias and leukemias is based largely on the findings in 100 cases which may be classified as follows:

Primary Anemia	12
Secondary Anemia from Acute Blood-loss	28
Secondary Anemia from Other Causes:	
Cancer	6
Septic Processes	5
Ulcerative Colitis and Other Bleeding from the Gastro-intestinal Tract	4
Uterine Bleeding (Not from Cancer)	8
Malnutrition in Infants	4
Diseases of Liver or Biliary System	3
Bleeding from Kidneys	7
Acute Lymphatic Leukemia	12
Lymphatic Leukemia, Known Duration over 2 Months	6
Banti's Disease	1
Purpura Hemorrhagica	4

In primary anemia, when the red-count was very low, (1,000,000 or less) hemorrhages were always present, usually small, scattered, streaked or blotchy, and did not obscure the vessels. In the more severe cases they were larger (often half the size of the disc) and also streaked or blotchy. Or they occurred as still larger patches with irregular outlines; these often were in the deeper layers of the retina, obscuring vessels. When a few days old these larger hemorrhages sometimes showed white, more or less glistening portions 1/10 to 1/8 of the whole in size. Such hemorrhages and the pre-retinal hemorrhages described in the second paragraph below may also be seen in leukemia.

During remissions in primary anemia, or when by treatment the blood is kept at a high level, the hemorrhages disappear and leave no visible trace. That the white areas seen in the larger hemorrhages are a part of the extravasated blood is manifested by their disappearance with the absorption of the hemorrhage.

In one case of primary anemia, moribund on admission to the hospital, at both maculae were large ovoid, dark, homogeneous, pre-retinal hemorrhages. In another severe case there were peri-papillary, streaked hemorrhages in both eyes.

Case 4. (Prov. Lying-in Hosp., 12489):

A woman of about 30 was admitted four days after the birth of her fifth child because of extreme weakness and anemia. Six months before she began to feel tired and short of breath on exertion; she also noticed that her color was gradually poorer. These symptoms continued and recently she has had a cough. At night she has felt as though she were "skating, riding or stringing blocks" and at times she would fail to recognize her husband; this was also at night. Her mouth has been sore for the last few weeks. The labor was normal and of short duration.

The blood: Hgb. 22%, R. B. C. 1,300,000, W. B. C. 12,600, P. 70%, L. 21%, Myels .9, Wass. neg.

The retinae were pale; the discs were blurred in outline and slightly elevated; there were large fan-shaped peri-papillary hemorrhages, streaked and extending for the most part along veins.

The patient's blood improved under treatment with liver; the hemorrhages gradually disappeared and 16 days after admission they had gone completely. The patient's blood then showed: Hgb. 57%, R. B. C. 3,900,000 and she was sent home.

No retinal changes except pallor were seen in the 28 cases of anemia from acute hemorrhage. In each of these cases the blood-loss was so great that transfusion was done and the retinae were examined before and within 24 hours after the transfusion.

Retinal hemorrhage in secondary anemia occurs only after the anemia has persisted for a comparatively long time. From my observations it would seem that an anemia, such as is produced from prolonged bleeding or repeated losses of blood, shows retinal hemorrhage only after an average hemoglobin-content of 50% has lasted for at least a month. Then, in some cases, a few small, streaked bleedings are seen along veins. The most extreme hemorrhages seen in this series, in cases of secondary anemia, were in a man who was known to have lost blood more or less continuously for 18 months, from a duodenal ulcer.

Case 5. (R. I. Hosp. 239047):

In this case, a man of 73, at the first observation the Hgb. was 18% (Sahli), the red count 1,100,100, and a few scattered streaked retinal hemorrhages were seen. After a stay of seven weeks in the hospital, with several transfusions, dietary and medicinal treatment, the Hgb. had risen to 70% and the red count to 4,200,000. The patient left the hospital, probably neglected all treatment and consumed much alcohol. On re-admission, five weeks after discharge, the Hgb. was 20% and the red count 1,000,000, the fundi showed large streaked hemorrhages, some six times the size of the disc in area, and irregularly shaped, white areas, which, like the hemorrhages, overlay the vessels. Clinically there was no nephritis. This patient died from perforation of a large, chronic, duodenal ulcer; autopsy, as well as complete blood study, confirmed the diagnosis of secondary anemia.

Vivoli of Florence (Revista di Clinica Medica, Florence 32:67 Jan. '31) from a study in 53 cases and from many others cited, concluded that retinal hemorrhages occur in 60 to 70% of cases of myelogenous leukemia and 40% of cases of lymphatic leukemia. He found that retinal or cerebral lesions (cerebral or meningeal hemorrhages in 2% of the cases) may occur before the general diagnosis is made.

In 2 of our 18 cases of leukemia there were scattered small hemorrhages with white areas suggest-

ing aggregations of white cells, fibrin, etc.; this is called leukemic retinitis. In others the first retinal changes seen were larger streaked hemorrhages, peri-papillary, or elsewhere in the retina. Either of these conditions may appear before there are any cutaneous or mucous membrane hemorrhages. In the acute cases (and the younger the individual the more acute and severe, apparently, is the disease) these hemorrhages were followed by larger, blotchy hemorrhages in the deeper layers of the retina. Six patients complained of poor vision. In these 18 cases of lymphatic leukemia, 10 of which had retinal hemorrhages, some of the smaller hemorrhages disappeared but the retina was never free from hemorrhages once they were present. After transfusion, in some cases, hemorrhage from the nose and mouth ceased for several days, hemorrhages into the retina, on the other hand, in at least three cases, followed transfusion within 24 hours.

In the most acute cases, when the patient's condition became very poor, large pre-retinal hemorrhages appeared, obscuring the entire macula in both eyes. In these cases finally the retina lost entirely its usual topography, the background consisted of wide stretches of orange-red color without apparent structure or defined limits, alternating with similar wide streaks of deep red color. These patients were too sick to complain much of poor vision which undoubtedly was present.

Case 6. (R. I. Hosp., 247777):

A man 18 years of age complained for four days of headache, nose-bleeding, hematuria and pain in the right flank.

There was much fresh blood in the urine, bleeding from the nose and gums and prostration. The blood:

Wass. and Hinton—neg., Hgb. 20% (Sahli), W. B. C. 5,050, R. B. C. 1,540,000, Coag. time 2½ mins., Bleeding time 12½ mins. White cells: P. M. N. 16, P. M. E. 1, P. M. B. 1, S. L. 68, L. L. 8, L. M. 1, Myelocytes 0, Lymphoblasts 3. Platelets—much diminished.

In both eyes there were flame-shaped hemorrhages, some large, about the discs and scattered less abundantly throughout the retinae.

This man had several transfusions. After each transfusion the bleeding from the nose and mouth would cease for four or five days, but at least twice fresh hemorrhages, all of the type described, appeared in the retinae.

The patient died 28 days after admission. Before death the white count had risen to 26,000, 90% of which were lymphocytes.

Case 7. (R. I. Hosp. 253036):

A married woman of 30 was admitted to the Gynecological department because of a foul vaginal discharge and general weakness. She had had two children, 8 and 6 years, and leucorrhea since the second child. In the last few weeks severe headaches, spells of dizziness and noises in the head.

Examination revealed a profuse mucopurulent discharge tinged with blood. Dr. Ira Noyes said, "The cervix looks necrotic, like the condition after deep cauterization."

The blood showed: Hgb. 30% (Sahli). R. B. C. 1,510,000. W. B. C. 1,550. Diff.: Polys. 30%. Lymphs, 70%. Wass. Pos. with cholesterin, Neg. with acetone. Hinton Neg. Blood Chem.: Urea 19 mgm.% Sugar 89 mgm.%. Platelets normal. Reticulated cells 0. Gastric analyses showed total acidities of 16 and 20.

Dr. H. A. Lawson, after studying a blood smear, felt that this was a case of aleukemic leukemia. Smear showed 90% lymphocytes, of which 10% were classified as lymphoblasts.

On the next day Dr. W. B. Castle examined a smear and pronounced it definitely not a case of pernicious anemia; reasons: "Red cells are too uniform in size and shape for primary anemia with a red count as low as 2,000,000; these are definitely abnormal white cells."

Bone marrow biopsy-report (Dr. B. E. Clarke): "Malignant lymphoma of the bone marrow."

Eye examination June 18: In the right eye—there was noted a blotchy, pre-retinal hemorrhage, three times the size of the disc, about 1½ disc-diameters from the disc, directly downward. In the left eye—three pre-retinal hemorrhages, each about the size of the disc, one at the macule, the others about one disc-diameter from the disc at 10 and 6 o'clock respectively.

Six days later, no new hemorrhages were to be seen, those described seemed slightly smaller than before.

The patient was discharged 38 days after admission. Hgb. 40%. R. B. C. 1,950,000. W. B. C. 4,000.

Diagnosis: Atypical Leukemia.

Case 8. (R. I. Hosp. 250,472.):

A married woman of 40 with the following history: "Had 'septic sore-throat' seven weeks ago; she was confined to bed four weeks and since she got up has had dizzy spells, prickling in the fingers, stiffness and swelling in her neck, and a 'weakness' in her eyes which she cannot explain. She talks volubly and has many complaints, indefinite as to detail.

"She is pale; the tonsils are rough but not inflamed; the knee-jerks are exaggerated; there is a double Babinski; otherwise the examination is negative. The spleen is not felt; no glandular swellings noted. The blood: Hgb. 30% (Sahli). W. B. C. 11,000. R. B. C. 1,600,000. Microcytes 100%. Reticulated cells 0.5%. Wass. neg."

Three days later: Hgb. 28% (Sahli). W. B. C. 10,000. R. B. C. 850,000. Platelets—relatively increased. Five days later, after blood transfusion: Hgb. 23% (S). W. B. C. 3,400. R. B. C. 1,340,000. Color index 0.9. P. M. N. 56. S. L. 44. Platelets normal. At this time the Benzidine test negative. There was free hydrochloric acid in the vomitus and the icteric index was 7.

The eyes were first examined after the transfusion on the day of this last-mentioned blood examination: Both retinae showed a large number of small hemorrhages, some with white centers, mostly in the region within three disc-diameters of the disc margin.

Dr. H. A. Lawson made a diagnosis of lymphatic leukemia. A piece of the sternum was removed and examined by Dr. B. E. Clarke who reported "Malignant lymphoma of the bone marrow."

Ten days after the first eye examination there were a very few small hemorrhages in the retinae; at the sites of many of the hemorrhages noted previously were small, pale blurred areas, faintly yellow in color. The patient had been seen daily and it seemed that no new hemorrhages appeared after the first examination.

By this time the spleen could be felt. A few swellings appeared in the axillae and groins. Death occurred 28 days after admission.

Case 9. (R. I. Hosp. 239,581):

A boy of 12 suffered for two weeks with headache and lassitude. Twenty-four hours before admission he vomited a large amount of blood; the vomiting recurred several times and he passed blood from the bowels.

The skin was pale; the spleen enlarged.

The blood: W. B. C. 12,100. R. B. C. 2,550,000. Hgb. 60%. P. M. N. 71 Lymph. Small and large—21. Unclassified 16. Wass. neg.

X-ray exam. of chest and G. I. tract neg.

No blood in the urine.

His ocular fundi were examined daily. The Hgb. fell to 20%, the white count rising to 14,000. He was given a blood transfusion four days after admission and a second nine days after admission. On the day following this transfusion for the first time there were splashy hemorrhages throughout the retinae except at the maculae. These gradually disappeared. Two successive transfusions were not followed by hemorrhage. The boy's general condition improved; a diagnosis of Banti's disease was made and splenectomy was done by Dr. Charles O. Cooke. Twenty days after this, 42 days after admission, he was discharged. The pathologist confirmed the diagnosis of Banti's disease. When he left the hospital the Hgb. 80%. R. B. C. 4,250,000. Eighteen months later, in good general condition: Hgb. 70%. R. B. C. 3,900,000.

Case 10. (R. I. Hosp., 249,165):

A man 27 years old, whose elder brother died of lymphatic leukemia at 26, and whose chief complaint was increasing weakness for four weeks, had bleeding from the nose, slight in amount but frequently recurring. His blood Hg. 28 (Sahli). R. B. C. 1,340,000; Mean. Corp. Vol. 1.3. Ret. Cells 5.8%. W. B. C. 60,600. Diff. count P. M. B. 19. S. L. 29. L. L. 45. Lymphoblasts 3. Myelocytes 3. Pre-myelocytes 1. There were no hemorrhages into the skin, no hematuria, no gross bleeding from the gastro-intestinal tract, benzidin test negative. During the night he had a convulsion, in the morning he noticed that he could not see with his right eye. The disc was somewhat elevated and completely blurred. There were a few small, streaked bleedings on the disc-swelling, several large peri-papillary hemorrhages. The retinal veins were large and the arteries small and pale. At the macule a hemorrhage about two-thirds the size of the disc, dark, homeogenous, and bulging forward. In the left eye, the disc was not elevated, its margins were distinct and no hemorrhage seen about the disc, near the macule or in the periphery. The clinical explanation given for the convulsion was that it was an ischemic reaction. There was no lumbar puncture. It seems possible that this may have been a case of cerebral or meningeal hemorrhage described by Vivoli (vide supra).

Comment: Papilledema in leukemia has been described, in some cases as secondary to the increased intra-cranial pressure accompanying it, in others, where increased intra-cranial pressure was not present, to lymphstasis. This case is interesting because of the sudden loss of vision in one eye after a cerebral attack and because the other eye showed no evidence of local or cerebral involvement.

The hemorrhages in the case of Banti's disease were large and streaked. This patient, a boy of 12, was given six blood transfusions, the hemorrhages appearing after the second of these. They disappeared in 14 days, in which time he had been given two more transfusions. Later the spleen was removed and he has remained clinically much improved for 18 months.

In three cases of hemorrhagic purpura there were large, streaked, retinal hemorrhages, followed by ovoid hemorrhages at the macule, with poor vision. In one case, where there were many petechiae in the skin but where the purpura was supposedly of the symptomatic type, there were no retinal changes seen.

Discussion: In the blood dyscrasias it seems probable that the most important factor in production of retinal changes is a change from normal in the composition of the blood. In general, hemorrhages are more prone to occur when the blood platelets are diminished but these may be low and no hemorrhages seen (at least in the retinae); and where they are relatively not lower than the other constituents and anemia has been present for a long time, as from prolonged bleeding from the intestine or uterus, retinal hemorrhage may occur.

The more acute in onset and rapid in progress are the changes in the blood, the more extensive and numerous are the retinal changes, especially hemorrhages. In the chronic, slowly progressing cases, hemorrhages are more rarely seen, and when seen are not large in size or number until late in the course of the disease.

Retinal hemorrhages in these anemic cases indicate to some extent the severity of the case; the type and extent of the hemorrhages vary according to the degree and the duration of the variation of the blood from the normal. That the variations of some particular element of the blood has specifically to do with the production of retinal bleeding, has not yet been shown. Retinal hemorrhages may be first evidence of the tendency to bleed in these cases. Absorption of the extravasated blood takes place with improvement in the blood state.

From observations in these 100 cases it seems that the condition of the walls of the small vessels

is an important immediate factor in the production of hemorrhage, since in the extreme anemias from acute hemorrhage retinal hemorrhages were not seen, but were seen in cases of prolonged, recurrent blood-loss where, presumably, the imperfect nutrition of the vessel-walls made them permeable. Also, in the occurrence of hemorrhages as in the case of Banti's disease, following transfusion; the blood given was compatible and did the boy good but some escaped through the vessel walls.

It does not seem possible to tell from the retinal appearances the type of blood disease present. Not only the exact nature of the disease but the degree and duration of abnormality of the blood modify the picture.

BIBLIOGRAPHY

Where direct reference has been made in the literature citation has been made in parenthesis.

This listing is supplementary.

Metabolism of Eye; Physiological Aspects. (Lucien Howe Memorial Lecture) Duke-Elder, W. S. Arch. Ophth. **6,** 1-20, Jul. 1931.

Neuro-retinitis Associated with Nephritis and Hypertension. J. Rollet and Panfique, Ann. d'ocul. **167,** 773, Sept. 1930.

The Differential Diagnosis of Tumor of the Brain; The Importance of Considering Renal Hypertension with Choked Disk. Grant, F. C. Arch. of Neur. and Psych. **27,** 4, 816.

Retinal Changes of Arterio Sclerotic Heart Disease and Essential Hypertension. Horine, E. F. and Weiss, M. M. Arch. Ophth. **6,** 533-543, Oct. '31.

Retinal Changes in Hypertension and Renal Disease. R. Floyd. Arch. Ophth. **6,** 433-444. Sept. '31.

Cases of Hypertension Studied with the Aid of the Ophthalmoscope. H. C. Messinger. R. I. Med. Jour. Jun. 1928.

Retinal Hemorrhages After Blood-transfusion. Messinger, H. C. and Eckstein, A. W. R. I. Med. Jour. Nov. 1931.

Ueber Leukaemische Fundusverandungen. Kreibig, W. **76,** 6, Mar. 1932.

Fundus Changes in Leukemia. Sven Larsson. Acta Ophth. **8,** *22,* 1930.

Ocular Findings in Diabetes. Shepardson, H. C. and Crawford, J. W. Cal. and West. Med. 1931 **35,** 11, Aug. 1931.

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